Ceptor Animal Health News

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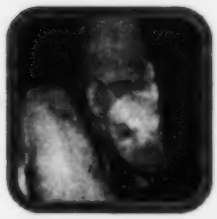

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Ministry of Agriculture, Food and Rural Affairs

Ontario

Acidifying Whole Milk and Milk Replacer with Citric Acid

Paisley Canning and Taryn McIntyre, OMAFRA Summer Experience Students, and Neil Anderson, Veterinary Science and Policy Unit, OMAFRA

Neither formic acid nor milk replacer powder (MR) has an approval for rearing milk-fed calves in organic dairy production. Citric acid does and thus there is interest in using it to acidify whole milk (WM) for free-access feeding systems. This report shows our findings related to acidification of bulk tank Jersey milk (4.7% fat, 3.7% protein) and a 20% protein, 17% fat all-milk-source milk replacer.

We acidified MR to a pH of 4.4 by adding 5 grams (g) of citric acid to 1 litre (L) of MR mixed at 150 g/L and WM to a pH of 4.3 by adding 5.8 g of citric acid to 1 L of WM. A level teaspoon of powdered citric acid weighed 6 g. Twice daily over 95 hr. we recorded pH, temperature and organo-leptic changes in control and acidified samples kept at room temperature (22.5-24.5°C). After doing the organo-leptic tests, we stirred samples prior to testing pH.

Figure 1 shows that the pH of the acidified WM remained fairly constant with a slight decrease at 48 hr. The pH of the control WM sample decreased at 30 hr. and continued to decline until the end of the trial.

WM separated minutes after initial mixing with citric acid. Although varying in amount, acidified WM separated into a whey-like fraction prior to each observation. Acidified WM smelled slightly sour compared to control WM but did not smell rancid until 81 hr. Control WM changed from a sweet to sour odour at 55 hrs.

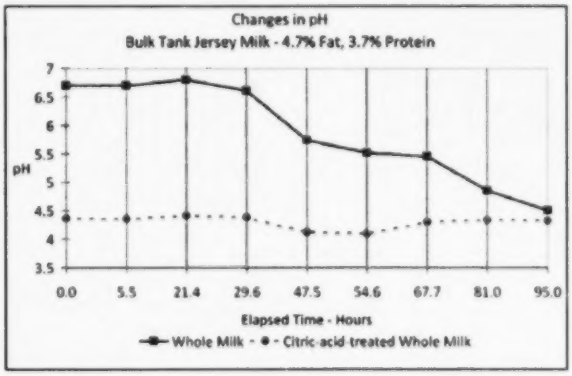

Figure 1. Changes in pH over 95 hr. for whole milk and citric-acid-treated whole milk kept at 22.5-24.5 °C.

Figure 2 shows that the pH of the acidified MR remained fairly constant until 55 hr. The pH of the control MR declined considerably between 30 hr. and 48 hr.

(Continued on page 3)

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Acidified MR samples separated about 10 minutes post acidification. We mixed the samples 5.5 hr. later and found no further separation at subsequent observations until 55 hr. Acidified MR samples started to smell unpleasant and sour at 85 hr. Control MR changed from a sweet to sour odour at 48 hrs.

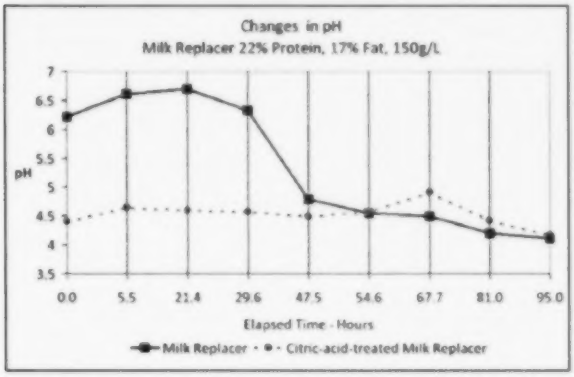

Figure 2. Changes in pH over 95 hr. for milk replacer and citric-acid-treated milk replacer kept at room temperature.

Whole milk acidified to a pH of 4.3 maintained a pH of 4.0 to 4.5 for 95 hr. Milk replacer acidified to a pH of 4.4 maintained a pH close to 4.5 for most of the observation period.

Figure 3 shows a visual comparison of separation at 48 hr. between acidified WM (top) and acidified MR (bottom). Note the liquid layer present in the acidified WM samples. Separation of citric-acid-treated WM may be problematic as clotted, curdled milk can plug lines and nipples on feeding systems.

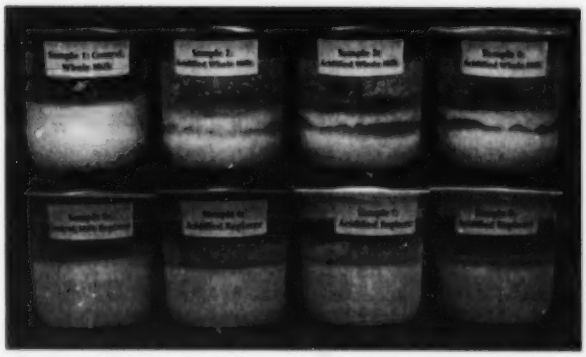

Figure 3. The top row shows photographs of control and citric-acid-treated whole milk samples and the bottom row shows photographs of control and citric-acid-treated milk replacer samples taken at 48 hr.

Lastly, we did a cost comparison using formic acid, Agri-ACID® and citric acid powder. As shown in **Table 1**, when citric acid is purchased in 25-kg packages, the cost to acidify 1 L of milk is similar for citric acid ($0.017) and formic acid ($0.018) but Agri-ACID® is more expensive ($0.040).

Based on pH and organo-leptic evaluations, citric acid may be an alternative preservative for WM or MR. However, from this experiment, we do not know if citric acid either inhibited bacterial growth or killed bacteria. Our tests did not evaluate the ability of citric acid to kill specific bacteria, e.g., *Escheria coli, Staphylococcus aurens, Mycobacterium avium paraTB (Johne's).*

Table 1. Comparison of Costs to Acidify Milk Using Three Acidifiers.

	Amount needed to acidify 1 L of milk replacer	Amount needed to acidify 1 L of whole milk	Cost per bag	Cost to acidify one litre of milk replacer	Cost to acidify one litre of whole milk
Citric Acid	5 g	5.8 g	25 kg for $72.20	$0.014	$0.017
			500 g for $6.50	$0.065	$0.075
Formic Acid	30 mL (1 part formic acid 85% into 9 parts water)	30 mL (1 part formic acid 85% into 9 parts water)	20 L for $119.95	$0.018	$0.018
Agri-ACID®	70 mL (1 part Agri-ACID® into 9 parts water)	70 mL (1 part Agri-ACID® into 9 parts water)	20 L for $119.95	$0.042	$0.042
			115 L for $665.00	$0.040	$0.040

Prices and quantities available from Farmers Farmacy, *www.farmersfarmacy.com*, August 6th 2009

Neck Rails With and Without Nylon Straps

Neil Anderson, Veterinary Science and Policy Unit, OMAFRA, and Paisley Canning, OMAFRA Summer Experience Student

Nylon straps installed tailward of a neck rail may cushion trauma associated with neck to neck-rail contact during rising motions. In effect, the nylon straps are a secondary *neck rail* in the stall. When some of the nylon straps broke at Scenic Holsteins, the owners offered us the opportunity to study free-stall usage in a pen having stalls with and without a nylon strap. We are unaware of research related to nylon straps. Our findings may be of use to you and cows in your care.

Our observations were made in a slatted-floor barn in a single pen with 74 tail-to-tail stalls and 91 cows (123% occupancy). The cameras focused on stalls facing the feed alley. Our data came from eight stalls with a neck rail (pipe) and ten stalls with a neck rail plus a nylon strap (**Figure 1**). The neck rail was mounted 70 inches forward of the alley curb and the nylon strap was 62 inches forward of the curb (horizontal dimension). The bottom of the neck rail and the nylon strap was 50 inches above the stall surface.

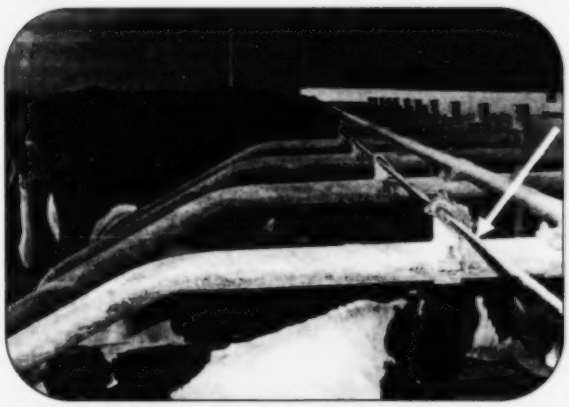

Figure 1. The neck rail and nylon strap are mounted to the top pipe of the loop dividing stalls. (Arrow points to the nylon strap.) Another section of stalls had the nylon strap removed.

We collected data using time-lapse video between noon on May 12 and mid-morning on May 16, 2009. At ten-minute intervals, we recorded stall usage in each stall as: empty, perching with two feet in the stall, standing with four feet in the stall or resting (lying). In **Figure 2**, we have summarized stall usage data as proportions of total observations. We tested the hypothesis that stall-usage proportions without the nylon strap would equal those with the nylon strap. We found no significant difference for empty (0.24 vs. 0.23), standing (0.10 vs. 0.09) or resting (0.60 vs. 0.60). Although perching was infrequent, it was significantly greater in stalls with the nylon strap (0.067) than in stalls without the nylon strap (0.056) ($Z=2.2$, $P=0.02$, 95% CI= (0.00165 - 0.02038)).

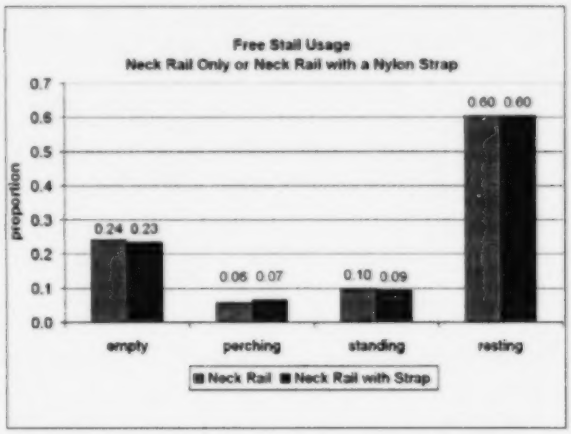

Figure 2. The proportion of observations with stalls empty or stalls used for perching, standing or resting in free stalls without and with a nylon strap associated with the neck rail.

The owners of Scenic Holsteins installed nylon straps for welfare reasons. When installed, little if any information would have been available about cow comfort related to neck rails. Now we are learning that the placement of neck rails may contribute to lameness. Florian Bernardi (J Dairy Sci 2009, 92:7; 3074-3080) reported his research cows spent more time perching when housed in pens with more restrictive neck rails (52 inches from the curb vs. 76 inches). He also showed that new cases of lameness and sole lesions were more frequent when his cows were housed in pens with restrictive neck-rail placement. Interestingly, we found a difference in perching even with the nylon strap restricting the space by only eight inches (strap at 62 inches and neck rail at 70 inches).

(Continued on page 5)

The nylon strap at Scenic Holsteins poses a predicament; it prevents contact with the metal neck rail but it may contribute to lameness through perching. As a compromise, let's consider moderating the strap's effect on perching by moving it to a typical neck-rail location, 68-70 inches forward of the alley curb, and moving the metal neck rail ahead of it. The new location may maintain desirable cushioning effects while diminishing undesirable perching. Some additional on-farm research is needed to determine if the metal neck rail is necessary.

Acknowledgement. We are grateful to Stefan Weber and his family, Scenic Holsteins, St. Mary's, Ontario, for their kind invitation to conduct this study and their permission to share the findings.

Staph aureus Mastitis – Finding the Treatable Cow

Ann Godkin, Veterinary Science and Policy Unit, OMAFRA

Staphylococcus aureus (*SA*) remains a common cause of persistent mastitis in most Ontario dairy herds. Successful control programs are the desire of both herd owners and their veterinarians.

Monitoring the pattern of *SA* mastitis over time remains difficult but essential for the fine tuning of ongoing prevention programs. Rapid and accurate identification of *SA* infection can be achieved by testing preserved cow test-day samples from milk recording using the PCR test, or by testing aseptically collected teat-end milk samples by bacteriological culture.

Therapy, if successful, can shorten the duration of infection and reduce the risk of infection spreading to other cows, but has not often been utilized in this manner in *SA* mastitis prevention programs because it is notoriously unsuccessful and expensive. Cure rates reported in research are highly variable; for subclinical *SA* mastitis, cure rates reported range from 4 to 92% (as cited by Barkema). Numerous factors are known to impact cure rates and affect therapy outcomes. Such factors include cow parity, days in milk (DIM), front vs. rear quarters, somatic cell count (SCC) at treatment time, duration of infection, number of colony forming units (CFU) on culture, number of quarters of the cow infected and duration of follow-up. Utilizing all this information in a practical sense has proven difficult. Beyond these oft-studied factors, recent research with advanced microbiological techniques is revealing that differences in strains of *SA* may also interact to impact on treatment success on the farm and may help to explain the disparity in research results.

For herds with cows infected with *SA*, the goal should be to make use of all available information to develop a standard approach to therapy decisions. Historically, one approach has been to attempt at least one treatment of most cows with mastitis. The current approach for *SA* mastitis is for herd owners to resist treatment except for an exceptional, treatable, cow. They need to identify the right cow, at the right stage of infection, infected with a potentially responsive strain of *SA*. Cow and herd data can be used to develop treatment algorithms for farms to select cows that are "treatable" - for example, "young" cows, "early" in infection and infected with a susceptible strain. The definitions of "young" and "early" may be herd specific and will vary depending on overall infection patterns, infection pressure and economics. Defining "susceptible" strains, however, is key. New information is helping us define these strains.

Disc assay antimicrobial susceptibility testing of *SA* isolates from milk cultures has little ability to predict mastitis cure rates, and is disappearing from laboratory services offered around the world. Classifying strains of *SA* as penicillin-susceptible or resistant based on β-lactamase production has emerged as a much more useful service. Over the last few years the Animal Health Laboratory in Guelph has offered testing of *SA* isolates for β-lactamase production, and within a few months the PCR testing on DHI samples will provide detection of the βlaZ gene in *SA* isolates. *SA* strains that have the βlaZ gene can produce β-lactamase, which inactivates penicillin, therefore identifying strains that are resistant to penicillin-containing mastitis treatments.

(Continued on page 6)

Most surveys of *SA* for antibiotic resistance using MIC testing continue to show that most mastitis isolates, while often resistant to penicillin, still remain highly sensitive to most other antibiotics. In spite of this demonstrated in vitro susceptibility, cure rates for therapy of both clinical and subclinical *SA* mastitis remain low even when antibiotics in non-β-lactam classes are used for mastitis therapy (trials cited in Barkema, *et al*).

Interestingly, researchers have now shown that β-lactamase production not only predicts penicillin response but appears to correlate in a broader fashion to overall antibiotic susceptibility. When non-β-lactam antibiotics are used for treatment, the probability of a cure, even with antibiotics other than penicillin, is still lower for penicillin-resistant strains than those found to be penicillin sensitive.

The implication of penicillin-susceptibility testing is therefore broader than many may have assumed—for practical purposes, it appears that *SA* strains found to be β-lactamase producers are not only resistant to penicillin but often will not respond to therapy with other commonly available non-β-lactam antibiotics. Therefore, β-lactamase or βlaZ positive strains are not likely to be the "right" strains to treat with antibiotics.

Why this broad resistance occurs is not known, although Barkema and colleagues hypothesize that the gene for penicillin resistance may be one of several resistance genes that cluster in pathogenicity islands in certain strains of *SA*. If this proves accurate, then the use of a variety of antibiotics for treatment of penicillin-resistant *SA* strains may potentially select for strains of *SA* that are resistant to multiple antibiotics. This selection may contribute to the prevalence of such resistant strains within herds and across geographic regions.

The take-home message: *SA* mastitis strains should be tested for βlaZ gene (PCR) or for β-lactamase production (disc method). Finding a penicillin-resistant strain does not send the message "pick another drug", it means this is a mastitis case that is unlikely to respond to any antibiotic therapy. These are not the "treatable" cows!

For a good comprehensive review of therapy and *Staph aureus*, check out:

Barkema HW, Schukken YH, Zadoks RN. The role of the cow, pathogen, and treatment regimen in the therapeutic success of bovine Staphylococcus aureus mastitis. J. Dairy Sci 2006; 89:1877-1895.

Profit Profiler Dairy Financial Analysis Service from CanWest DHI Shows Wide Variation in Costs

W.J. (Bill) Grexton, Manager, Herd Management Services, CanWest DHI

The Profit Profiler service has been available to all dairymen since late fall 2008 and to date almost 80 dairymen have had their dairy business evaluated using the program. As of September 1, 2009, four companies and two individuals have been certified to offer it to customers, with two more waiting to be certified.

Profit Profiler Dairy Financial Analysis service is a financial, cropping, labour and production analysis tool that allows dairymen to identify bottlenecks with the goal of improving the farm's profitability. The service will evaluate a period of one year that corresponds with their financial fiscal year end. Projections will use that base and show the impact of change for another year. It will allow them to distinguish the measures that are common to progressive profitable dairymen.

Of the last 32 herds evaluated (with a 2008 year end), some interesting information is emerging. The average Milk Cow Feed cost (including home grown feeds) is 24% of milk revenue (compared to health and breeding at 3.4%). The range from the 25th to the 90th percentile is 17% to 28%, a relatively narrow range—but, for a 100-cow herd, it amounts to a difference of $65,550. Of this, the cost to grow forages averages 7.7% of milk revenue with a range of almost $20,000 (5.4% to 8.8% of revenue).

The replacement costs average 11% of milk revenue with the range of 7% to 12% (or another $27,000). The overall cost to produce $1.00 of revenue ranges from $.70 to $.85. For a $575,000 gross revenue, this difference amounts to $86,250 additional profit.

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There is tremendous potential to improve a producer's bottom line, even if he is in the 90th percentile. Anything we can do to help them identify the opportunities and make decisions to improve profitability only has a positive outcome.

This program can be useful for your clients. If you are interested in being certified to be able to offer it directly to your clients, you can contact Bill Grexton at CanWestDHI 1-800-549-4373 ext. 254. Bill is also available to speak at client meetings about the program and results.

More Information can be found at the website *www.canwestdhi.com/profiler.htm*

Lameness in Sows—A Problem in Ontario?

Tim Blackwell, Veterinary Science and Policy Unit, OMAFRA, and Paisley Canning and Taryn McIntyre, OMAFRA Summer Experience Students

Over the last several years, lameness in sows has been identified by some researchers in the U.S. as a welfare concern, as well as a problem leading to significant production losses in sow herds. Personal observations and casual discussions with swine practitioners in Ontario do not support the idea that lameness is a common problem on Ontario swine farms.

In an effort to support these observations with some standardized clinical observations, a trial is underway to record the occurrence of lameness in sows. Sows are to be scored by the stockperson both when they enter and leave the farrowing area. Sows showing no signs of lameness are scored a zero, slightly lame sows are a score of one and clearly lame sows are a score two. Scores are recorded on a single sheet that is faxed weekly to this office or can be sent by e-mail. A short DVD has been produced to help standardize these classifications.

This study does not attempt to distinguish between specific types or causes of lameness as this is often a difficult diagnostic undertaking. However if lameness in sows is an issue on specific farms, further diagnostic work will be undertaken. This study is open to all farms that have sows on site. It is not targeted only towards farms with lameness problems but such farms are encouraged to enroll. It is estimated that to record and submit the lameness data for this project will require between five and ten minutes weekly.

The primary goal of this project is to provide baseline values on the prevalence and severity of lameness in sows on Ontario swine farms. There will be one to two meetings for enrolled producers along with their veterinarians during the winter and spring of 2010 to discuss the results of the study along with possible causes, treatments and preventative practices for the lameness problems identified.

A small honorarium is offered to any producer interested in recording data for the 6-month study period. If you know of any producers who would be interested in contributing to this study, please contact Tim Blackwell at (519) 846-3413 or tim.blackwell@ontario.ca

Group Sow Gestation Pens: There is More to the Design than Square Footage Per Sow

Kathy Zurbrigg, Veterinary Science and Policy Unit, OMAFRA, and Franklin Kains, Engineering Consultant, Kitchener, Ontario

If sows need to compete for feed in a pen, aggressive encounters occur to gain access to feed. To decrease the severity and frequency of aggressive encounters, sows must be convinced that feed is not a limited resource. This is best achieved by feeding frequently and spreading feed out over a large feeding area. Distributing feed in numerous locations throughout a complex or structured pen can also eliminate aggressive encounters between sows.

How a pen is structured and how feed is delivered is critical to the success of pen gestation. Separate feeding areas within a single pen can be created with the addition of partition walls at the side or in the centre of the pen **(Figures 1 and 2)**. These partitions can provide timid sows with a place to escape from aggressive encounters and discourage aggressive sows from pursuit. A centre dunging alley is another way to structure a pen into distinct areas for different groups of sows to eat and rest. A third method of adding structure to a pen is to increase the number of sows in the pen. In groups of 20 or more sows, the sows themselves serve as partitions and create complexity within the pen.

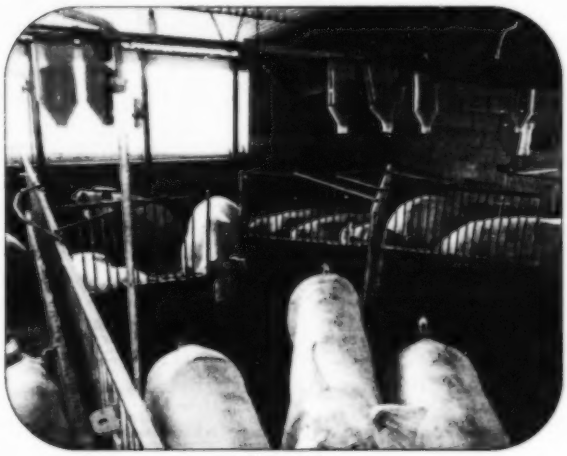

Figure 1

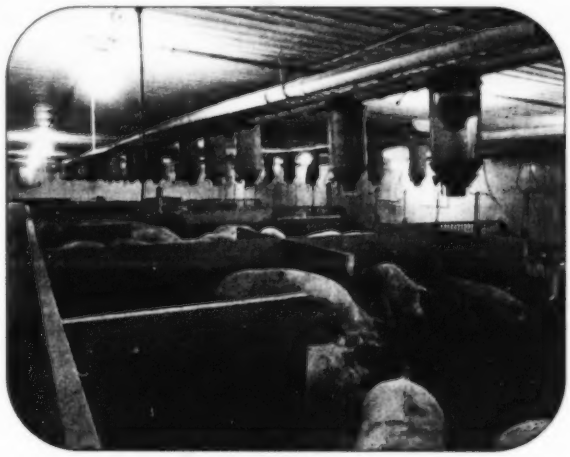

Figure 2

Partitions can provide timid sows with an escape from aggressive encounters and discourage aggressive sows from pursuit.

Where partially-slatted finishing pens are to be converted into gestation pens, the partitions over the dunging area between two or three pens should be removed. The solid-floored portion of each pen then becomes a separate feeding area for drop feeding while the remaining interior walls serve as partitions (**Figure 3**). This increases the total number of sows per pen because each pen is now larger. These additional sows add complexity to the pen and the sows benefit from the increased "shared" space (**Figure 4**).

The "right" number of sows per pen is determined in part by the number of farrowings per week. Farms with less than 15 farrowings per week should consider batch farrowing so that a larger number of sows can be grouped in a pen. Some producers choose to have a "dynamic" pen, where sows are continually added and removed. Pen design, space allowance per sow and feeding methods are much more critical in a dynamic pen. Sow aggression is more likely to be continuous in a dynamic pen system as new fights may ensue each time sows are added or removed.

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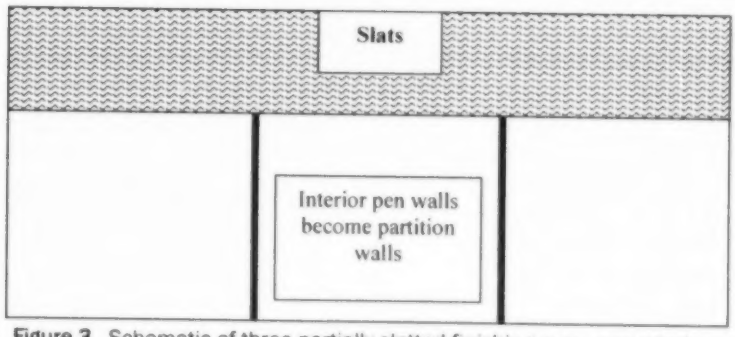

Figure 3. Schematic of three partially-slatted finishing pens converted into one sow gestation pen by removing the partitions between the pens over the dunging area.

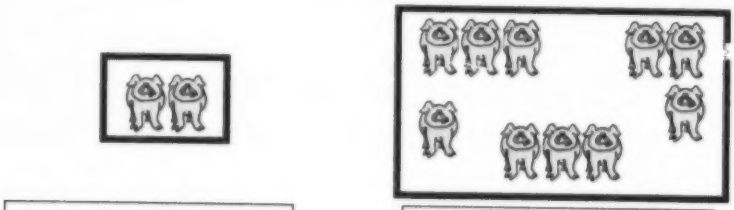

Figure 4. Diagram of increased "shared space" per sow.

Farms farrowing more than 40 sows a week should have two sow pens for each week so that sows can be divided into the two pens by size and/or body condition. This allows the producer to better control the amount of feed per sow, decreasing feed to the pen of larger sows and increasing feed to the pen of thin or smaller sows.

Peak efficiency with floor feeding is achieved by spreading out the feed in both time and space. Feed can be dispensed over the entire solid floor area by using cones or "y" diverters under feed drops. Feed can be spread out in time by feeding multiple times a day. The appropriate amount of ration for a pen of sows should be dropped in small amounts three to eight times throughout the day. This method prevents sows from becoming overly hungry and anxious at feeding time. A timer added to the auger ensures regular drops without added labour.

The producers that are using group gestational housing systems wean their sows into breeding stalls. If weaned into pens, sows can injure themselves or others when demonstrating estrus behaviour. The length of time the sows spend in the breeding stalls varies from farm to farm. Some producers mix sows into a pen immediately after breeding. On these farms, pregnancy diagnosis by ultrasound is done in the pen. Other producers prefer to hold sows in the breeding stalls for 35 days, until they are confirmed pregnant. If the gestation pens are well designed and managed, sows can be mixed immediately after breeding with little fear of aggressive encounters.

The housing designs and management systems described in this article were created on Ontario farms by producers who converted to group sow gestational housing. Swine producers created these systems through trial and error until they had limited or eliminated aggression between sows. The success of these systems has been demonstrated to their owners in a number of ways:

1. Sows have fewer scratches, injuries, and lameness's and their body condition remains consistent;
2. Farrowing rates are greater than 80%, with a few herds approaching 90%;

(Continued on page 10)

3. Producers rarely have to remove a sow that is not doing well in the group situation; and
4. Producers find working in group sow housing barns a quiet and pleasant alternative to crated gestation.

A new DVD showcasing four Ontario farms using group gestation housing is now available at no cost through the Ontario Pork Producers Marketing Board and the Ontario Ministry of Agriculture, Food and Rural Affairs (OMAFRA). The DVD includes a handout detailing the layout and management techniques of each farm that are discussed in the video. An earlier DVD (produced in 2002) is also available, which features three other group gestation housing barns in Ontario, including one that uses straw.

To receive either or both DVDs, go to *www.thepigsite.com/focus/5m/3785/ontario-pork-making-the-switch-to-group-housing-videos* or contact:

Kathy Zurbrigg, OMAFRA (519) 846-3418 or kathy.zurbrigg@ontario.ca

If you have questions regarding group housing or converting a conventional crated barn to group pens, please feel free to contact me.

Evaluation of the Prevalence of Drench Failure and Anthelmintic Resistance on Ontario Sheep Farms – Pilot Study

Janessa Wood, Evan Hanna, Andrew Peregrine and Paula Menzies, Ontario Veterinary College, and Jocelyn Jansen, Veterinary Science and Policy Unit, OMAFRA

Gastrointestinal nematode (GIN) parasitism is a severe and increasingly important production-limiting disease on sheep flocks worldwide, which also has implications for animal welfare [1,2,3]. In 2007, anthelmintic resistance (AR) was documented, for the first time, on a farm in northern Ontario. However, since no other farms in Ontario have been evaluated, the prevalence of AR is unknown. AR has a major impact on the profitability of the sheep industry elsewhere in the world and it is important to confirm the presence of AR in Ontario before it becomes widespread and common.

In this study, 17 farms were recruited by flock veterinarians that attended the Small Ruminant Veterinarians of Ontario meeting held on May 13, 2009. Participating farms were visited and four-gram fecal samples were collected from 15 individual lambs that had been pastured for a minimum of four weeks. Samples were individually analyzed using a modified McMaster technique to determine the number of eggs per gram (epg) of feces. Farms that had a mean fecal egg count of $\geq$ 200 epg were sent ivermectin to treat all their lambs. Fourteen days after treatment, producers were asked to collect fecal samples from 15 lambs for a "drench check". If drench failure occurred (mean reduction in egg count < 90%), an on-farm Fecal Egg Count Reduction Test (FECRT) was performed by the project team. This test was carried out by randomly allocating lambs into groups of 15 animals each and individually weighing each animal to ensure correct dosing of drugs. If numbers permitted, lambs were allocated to four groups:

1. Placebo (control),
2. Ivermectin drench,
3. Fenbendazole drench, and
4. Levamisole drench.

The results from this work will identify whether the drench failure was due to AR. The results will also show which drugs are efficacious, and to which drugs there is AR.

To date, 15 of the 17 farms showed levels of epg output that could be associated with an impact of infection on sheep health (i.e. fecal egg counts (FECs) > 200 epg). On the 15 farms that had high FECs and a drench check was performed, ten farms were identified as having drench failure (i.e. mean reduction in egg count < 90%). There are a number of reasons for drench failure occurring, and it

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should therefore not automatically be diagnosed as AR. Reasons include:

1. incorrect dosing due to technique;
2. incorrect dosing due to method of weight determination,
3. incorrect dosing due to mechanical issues with the delivery system (i.e. drenching gun), or
4. an animal in the flock was missed during drenching.

To date, FECRTs have been started on three farms. The FECRT will determine whether drench failure was truly due to AR or due to other reasons, as well as determine the susceptibility of GINs on-farm to the different classes of anthelmintic drugs. The FEC and FECRT data collected by this project, in conjunction with information on risk factors for AR, will allow for the development of management strategies that minimize the impact and spread of anthelmintic-resistant parasites in Ontario flocks.

This project is a pilot project for a larger project taking place over the next two years. We are looking for seventy flocks where the owner pastures ewes and/or lambs during the grazing season.

Please contact Dr. Andrew Peregrine at the Ontario Veterinary College (aperegri@ovc.uoguelph.ca) to nominate flocks.

1. *Wolstenholme AJ, Fairweather I, Prichard R, von Samson Himmelstjerna G, Sangster NC. Drug resistance in veterinary helminths. Trends in Parasitology 2004; 20:469-476.*
2. *Kaplan RN. Drug resistance in nematodes of veterinary importance: a status report. Trends in Parasitology 2004; 20:477-481.*
3. *Papadopolous E. Anthelmintic resistance in sheep nematodes. Small Ruminant Research 2008; 676:99-103.*

Equine Protozoal Myeloencephalitis (EPM): Laboratory Results for 2008 and 2009 and a Review of the Disease

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Excerpted from:

- the American Association of Equine Practitioners (AAEP) 2008 proceedings, A review of neurological diseases affecting horses, by Dr. Stephan Reed, and
- the American College of Veterinary Internal Medicine (ACVIM) Consensus Statement on EPM, J Vet Intern Med 2002;16:618-621.

Introduction

Sarcocystis neurona is the most common causative organism of Equine Protozoal Myeloencephalitis (EPM). *Neospora hughesi* can also cause the disease in rare cases. In the cycle of this disease, opossums excrete oocysts in stool, which develop into infective sporocysts in the environment. Intermediate hosts, including cats, skunks, raccoons and cowbirds, ingest the sporocysts where they develop into sarcocysts in muscle tissue. The life cycle is completed when opossums eat infected intermediary hosts.

EPM is a challenge for veterinarians to diagnose. There is no definitive test to diagnose EPM in affected horses. Clinical signs, cerebrospinal fluid (CSF) and serological testing, CSF cytological evaluation, response to treatment, and post-mortem examination are all used to attempt to confirm the diagnosis of EPM in a horse.

Clinical Signs

On physical examination of suspected cases, vital signs are usually normal. Most horses affected with EPM are bright and alert. Some horses appear thin and mildly depressed. Neurological examination often reveals an asymmetric weakness, ataxia, and spasticity involving all four limbs. Frequently, areas of hypoalgesia or complete sensory loss may be noted. The most frequent brain or cranial nerve deficits observed in horses at one teaching hospital included head tilt, depression, facial nerve paralysis, and difficulty swallowing.

The onset of clinical signs may vary from acute to insidious and include focal or multifocal signs of neurological disease. Early clinical signs of stumbling and frequent interference are easily confused with lameness. Horses with EPM often have asymmetric gait deficits with focal muscle atrophy. This may help distinguish EPM from other

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neurological diseases. Some horses affected with EPM may initially show abnormal upper airway function, unusual or atypical lameness, or even seizures. In severe cases, horses have difficulty standing, walking, or even swallowing.

In many horses there is a gradual progression of clinical signs, including ataxia, but in some horses mild clinical signs initially may be followed by a rapidly progressive clinical course. In other cases, the disease seems to stabilize or remain static for a time period. The variability of clinical signs occurs because the organism may attack randomly within the white and gray matter of the brain, brainstem, or spinal cord of the horse.

Laboratory Diagnoses

The Animal Health Laboratory (AHL), University of Guelph, sends all CSF and serum samples from suspected EPM cases to Cornell University for testing. AHL test information is summarized in **Table 1**. No post-mortem results were available to match with these test results to determine the sensitivity and specificity of these tests. Most practitioners submitted only serum for testing.

Serum is tested using the Western Blot (WB) test, which detects only anti-*Sarcocystis neurona* IgG. Since EPM only develops when the parasite enters the central nervous system (CNS), a positive serum test alone does not confirm EPM as the cause of neurological disease. This only confirms the horse has been exposed to *S. neurona*. In previous U.S. studies, seropositivity on the EPM WB test ranged from 33-53% in states where opossums were present. While exposure is common, development of clinical disease is rare.

When the parasite invades the CNS, antibodies can potentially also be detected in CSF. A positive CSF WB result provides support of EPM as the cause of the neurological disease. Care must be taken at collection to ensure that the CSF sample is not contaminated with blood. A red blood cell (RBC) count of CSF should be done within 1-2 hours of CSF collection, to prove that the sample is unlikely to be contaminated with peripheral blood. Samples that are grossly discoloured pink to red should not be submitted for Western Blot testing. At present, it is suggested that samples have no more than 50 RBCs/uL to be submitted for the Western Blot EPM test.

Serum or CSF WB test results are classified according to the strength of the antibody titre detected. Test results weaker than "positive" can be difficult to interpret. Samples with "weak positive" results may alert the clinician to a degree of reactivity that could indicate the presence of anti-*S. neurona* IgG. Because some horses might not develop a vigorous antibody response to *S. neurona*, these results could be consistent with evidence of EPM in some horses. This level of titre has been reported from samples collected shortly after experimental exposure. "Weak" reactions are borderline and should be interpreted as preliminary positives and confirmed with repeat sampling and testing in 3 to 4 weeks. A "low positive" represents what is believed to be a truly positive reaction.

The positive predictive value (PPV) of a test is defined as the probability that a horse with a positive test result truly has the disease. The WB on CSF has been shown to have a high PPV (90%) when used on a population of horses with horses with clinical signs of EPM, but a very low PPV (8%) when used on clinically normal horses. For this reason the test is not a valid method of screening horses for EPM on a pre-purchase exam.

A polymerase chain reaction (PCR) test for detection of *S. neurona* DNA in CSF is also available. Although a powerful and highly specific test, it has not been found to be clinically useful because of the many false negative results. This may be because parasite DNA is rapidly destroyed in the CSF environment or the parasite DNA is rarely present in the CSF. The American College of Veterinary Internal Medicine (ACVIM) states that PCR testing of CSF is of little value and it is not recommended for routine diagnosis of EPM.

In conclusion, a diagnosis of EPM is best established in horses that have clinical signs consistent with EPM and that have a positive WB test on an uncontaminated CSF sample. A favourable response to treatment, especially when subsequently followed by a relapse of similar clinical signs, is also supportive of a diagnosis of EPM in a live horse.

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Definitive diagnosis can only be made by post-mortem examination. PM results can remain inconclusive unless the parasite is detected on histological sections or is identified in tissue by immunohistochemistry or PCR.

Table 1. EPM Testing Results from the Animal Health Laboratory for 2008 and 2009 (to July 31)

Year	Number of horses tested	Serum WB only	CSF WB only	CSF WB & PCR	Tested both CSF (WB and PCR) and Serum (WB)
2008	55	51 Neg = 19 WP = 8 LP = 15 P = 9	2 Neg = 1 WP = 1 LP = 0 P = 0	1 WB = P PCR = Neg	1 Serum WB = positive CSF WB & PCR = Neg
2009	35	29 Neg = 11 Suspect = 1 WP = 8 LP = 5 P = 4	1 Neg = 1 WP = 0 LP = 0 P = 0	5 1 case LP for both tests, 4 cases Neg for both tests	0

Neg = negative, WP = weak positive, LP = low positive, P = positive
WB = Western Blot *S. neurona* IgG test at Cornell
CSF = cerebrospinal fluid

West Nile Virus National Surveillance Report— August 9, 2009 to August 15, 2009 (Week 32)

Kathy Zurbrigg, Veterinary Science and Policy Unit, OMAFRA

Canada

Human

No human cases of West Nile virus (WNV) infection have been reported to the Public Health Agency of Canada (PHAC) since the start of this 2009 WNV season.

Bird

As of week 32, a total of 187 dead birds have been tested by the Canadian Cooperative Wildlife Health Centre (CCWHC) since June 12, 2009, of which two (1.07%) were positive for WNV. The two positive birds were reported in Ontario (Middlesex-London and Wellington-Dufferin-Guelph). Both positive birds were American Crows.

Mosquito

As of week 32, eleven (11) mosquito pools positive for WNV have been reported in Saskatchewan.

Domestic Animal

One horse with WNV infection has been reported to the Canadian Food Inspection Agency (CFIA) since the start of this season.

National WNV surveillance data and maps can be found on the PHAC website at:
www.phac-aspc.gc.ca/wnv-vwn/index.html

Equine WNV data and maps can be found on the OMAFRA website at:
www.omafra.gov.on.ca/english/livestock/horses/westnile.htm

Provinces/Territories: Detailed WNV information can be accessed through the 'Links' section on the PHAC WNV website.

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United States

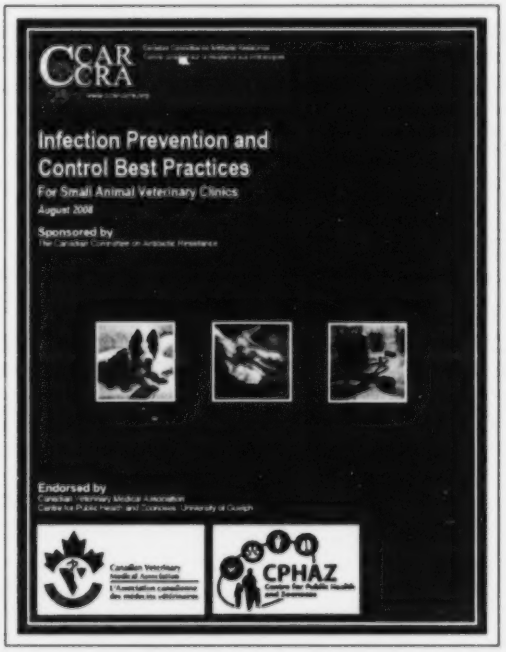
Human

As of August 18, 2009, eighty-two human WNV clinical cases [Arizona (7), Arkansas (1), California (10), Colorado (9), Idaho (1), Iowa (1), Kansas (2), Louisiana (3), Minnesota (1), Mississippi (15), Missouri (1), Montana (1), Nebraska (1), Nevada (10), New York (1), Pennsylvania (1), South Dakota (3), Tennessee (1), Texas(11) and Wyoming (2)] and twenty-seven presumptively viremic blood donors have been reported to the Centers for Disease Control and Prevention (CDC) in the U.S..

Of the 82 cases, 52 (63%) were reported as West Nile meningitis or encephalitis (neuro-invasive disease), 28 (34%) were reported as West Nile fever (milder disease), and 2 (2%) were clinically unspecified at this time. Of the 82 cases, there have been three deaths reported.

Detailed State information can be accessed through the CDC website: *www.cdc.gov/ncidod/dvbid/westnile/surv&control.htm*

Available Resources



Infection Prevention and Control Best Practices For Small Animal Veterinary Clinics

This free resource is relevant to mixed animal practitioners.

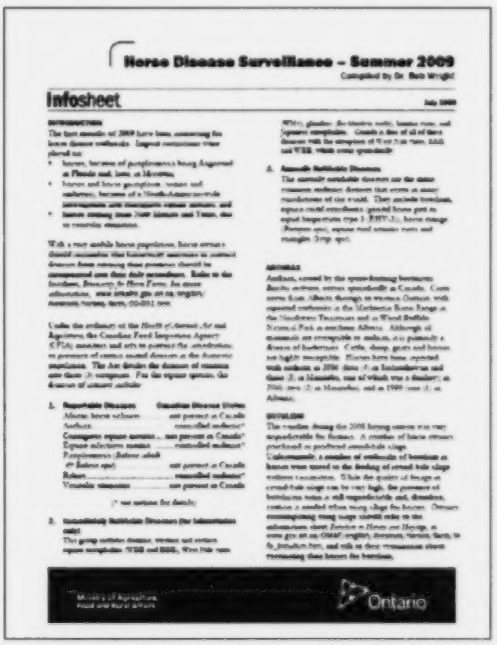
Download the PDF version at *www.ovc.uoguelph.ca/cphaz/resources/documents/GuidelinesFINALInfectionPreventionDec2008.pdf*

Horse Disease Surveillance—Summer 2009

The first months of 2009 were interesting for horse disease outbreaks. Import restrictions were placed on horses and horse germplasm.

With a very mobile horse population, horse owners should be advised to incorporate biosecurity measures into their daily procedures to prevent diseases from entering their premises.

Refer to the new information sheet at *www.omafra.gov.on.ca/english/livestock/horses/facts/info-surveillance_09.htm*


Horse Disease Surveillance – Summer 2009

Infosheet

Ontario

Continuing Education/Coming Events

October 1-4, 2009	New York State Veterinary Conference hosted by the New York State Veterinary Medical Society and the Cornell University College of Veterinary Medicine, College of Veterinary Medicine, Cornell University, Ithaca, New York. *www.nysvms.org*
October 10-17, 2009	American Dairy Goat Association Convention (American Association of Small Ruminants Practitioners' Program, October 11 & 12), Adam's Mark Hotel, Buffalo, New York. *2009adgaconvention.web.officelive.com/default.aspx*
October 13, 2009	Udder Health and Milk Quality on Organic Dairy Farms—a workshop presented by the Northeast Organic Farming Association of Vermont and the Vermont Veterinary Medical Association, Double Tree Hotel, South Burlington, Vermont. *www.vtvets.org/about/continuing_education.shtml*
October 14, 2009	Bovine Lameness Lecture and Wet Lab with Dr. Charles Guard, Animal Health Laboratory, Kemptville Campus, University of Guelph. Contact Jan Shapiro (613) 258-8320.
October 22 & 23, 2009	Central Canada Veterinary Association's fall conference, Strathmere Inn, North Gower, Ontario. *www.oavm.org (choose CCVA link)*
October 23 & 24, 2009	Ontario Association of Swine Veterinarians Fall Conference, Millcroft Inn, Caledon, Ontario. *www.oasv.ca*
November 5 & 6, 2009	17th Annual Swine Disease Conference for Swine Practitioners, Scheman Building, Iowa State University, Ames, Iowa. *www.ucs.iastate.edu/mnet/swinedisease/home.html*
November 12 & 13, 2009	Dairy Cattle Reproduction Council regional meeting, Crowne Plaza Riverfront, St. Paul, Minnesota. *www.dcrcouncil.org*
November 12-14, 2009	15th Great Lakes Dairy Sheep Symposium, Albany Marriott, Albany, New York. *www.dsana.org/symposia.html*
November 18 & 19, 2009	Ontario Association of Bovine Practitioners Continuing Education Program and Annual General Meeting, Holiday Inn, Guelph, Ontario. *www.oabp.ca*
November 19 & 20, 2009	Dairy Cattle Reproduction Council regional meeting, Doubletree Hotel Riverside, Boise, Idaho. *www.dcrcouncil.org*
December 4 & 5, 2009	2009 International PRRS Symposium: Molecular, Immunological, Genetic and Epidemiological Approaches for PRRSV Control, Downtown Chicago Marriott, Chicago, Illinois. *www.prrssymposium.org*
December 5-9, 2009	American Association of Equine Practitioners 55th Annual Convention, Mandalay Bay Hotel & Casino, Las Vegas, Nevada. *www.aaep.org/convention.htm*
January 28-30, 2010	Ontario Veterinary Medical Association Annual Conference, Westin Harbour Castle, Toronto, Ontario. *www.ovma.org/upcoming_events/conference.html*
January 31-February 3, 2010	National Mastitis Council's 49th Annual Meeting, Hyatt Regency, Albuquerque, New Mexico. *www.nmconline.org/meetings.html*
February 18-20, 2010	Ontario Association of Veterinary Technicians 32nd Annual Conference. *www.oavt.org*
March 6-9, 2010	American Association of Swine Veterinarians 41st Annual Meeting, Hilton Omaha Hotel, Omaha, Nebraska. *http://aasv.org/annmtg*
July 18-21, 2010	21st International Pig Veterinary Society Congress—Sharing Ideas-Advancing Pig Health, Vancouver Convention and Exhibition Centre, Vancouver, British Columbia. *www.ipvs2010.com*
November 14-18, 2010	26th World Buiatrics Congress, Espacio Riesco Convention Centre, Santiago, Chile. *www2.kenes.com/buiatrics2010/congress/Pages/General_Information.aspx*

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